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Attorneys for Defendant
CITY OF OAKLAND

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

SAN FRANCISCO DIVISION

OAKLAND BULK & OVERSIZED TERMINAL, LLC,

Plaintiff,

v.

CITY OF OAKLAND,

Defendant.

SIERRA CLUB and SAN FRANCISCO BAYKEEPER,

Defendants-Intervenors.

Case No. 3:16-cv-07014-VC

DECLARATION OF H. NADIA MOORE, PH.D., DABT, ERT IN SUPPORT OF DEFENDANT CITY OF OAKLAND'S MOTION FOR SUMMARY JUDGMENT, OR IN THE ALTERNATIVE PARTIAL SUMMARY JUDGMENT, AND OPPOSITION TO PLAINTIFF'S MOTION FOR SUMMARY JUDGMENT

Date: January 10, 2018
Time: 10:00 a.m.
Ctrm.: No. 2, 17th Floor
Judge: Honorable Vince Chhabria

I, Dr. H. Nadia Moore, Ph.D., DABT, ERT, hereby declare:

1. I am certified in toxicology as a Diplomate of the American Board of Toxicology and am admitted to both the United Kingdom and EUROTOX registries as a European Registered Toxicologist. I am a member of the Society of Toxicology, American College of Toxicology, British Toxicology Society, American College of Occupational and Environmental Medicine, American Association for the Advancement of Science, American Conference of Governmental Industrial Hygienists, American Chemical Society, American Industrial Hygiene Association, and Society for Experimental Biology and Medicine. I received a Bachelor of Science degree in Chemistry with a biochemistry emphasis from Pacific Lutheran University in 1992 and a Ph.D. in Environmental Toxicology from the University of Washington, School of Public Health and Community Medicine, Department of Environmental Health and Occupational Health Sciences in 2008. A true and correct copy of my full curriculum vitae is attached hereto as Exhibit 1. I have personal knowledge of the facts set forth in this declaration and, if called as a witness, could and would testify competently to such facts under oath.

2. I make this declaration in support of the Defendant City of Oakland's ("City") Motion for Summary Judgment, or in the Alternative Partial Summary Judgment, and Opposition to Plaintiff Oakland Bulk & Oversized Terminal, LLC's ("OBOT") Motion for Summary Judgment. Specifically, I address (1) OBOT's statement in its Brief (p. 34, lns. 3-6) that, "as the City's retained health expert in this litigation testified [referring to this Declarant], the U.S. EPA (which the City's expert concedes is both competent and capable) has identified 'permissible' concentrations of pollutants, including particulate matter, below which the 'health of any sensitive group of the population' will be protected;" (2) references in Declaration of Dr. Andrew Maier in Support of OBOT's Motion for Summary Judgment implying that there is no risk to human health if National Ambient Air Quality Standards ("NAAQS") are not exceeded; and (3) the "nonattainment" context under which ESA, in its report made part of the administrative record for the subject Ordinance, discussed NAAQS.

A. Increased Concentrations of Particulate Matter 2.5 Are Associated With Adverse Health Outcomes Among Exposed Populations.

3. Airborne particulate matter (PM) consists of a vast array of differently-sized particles. PM size categories correlate to the mass median aerodynamic diameter (MMAD) of the particles: PM_{10} corresponds to particles with MMADs less than or equal to 10 micrometers (µm) and $PM_{2.5}$ corresponds to particles with MMADs less than or equal to 2.5 µm.[1] Due to their smaller size compared to PM_{10}, $PM_{2.5}$ represents particles that are likely to be deposited in the alveolar zone of the lung if inhaled (where they may hinder gas-exchange function and/or may be directly absorbed into the bloodstream to exert potential systemic effects) and it was added as a criteria air pollutant to specifically represent the concentration of particles that penetrate more deeply into the lung.[2]

4. It is widely accepted within the scientific community that among exposed populations, increased concentrations of $PM_{2.5}$ pollution are associated with adverse health outcomes, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease.[3] Additional adverse health outcomes include nonfatal heart attacks and strokes, irregular heartbeat, increased risk of heart

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[1] California Air Resources Board. Overview: Diesel exhaust and health. Last updated April 12, 2016. Available from: https://www.arb.ca.gov/research/diesel/diesel-health.htm. Accessed: October 3, 2017.

[2] US EPA. Integrated Risk Information System (IRIS) Chemical Assessment Summary: Formaldehyde; CASRN 50-00-0. October 1, 1989., p.702; Maynard, R.L. Air Pollution. In: General and Applied Toxicology. 3rd ed. Ballantyne, B., Marrs, T.C., Syversen, T., editors. West Sussex, UK: John Wiley & Sons; 2009. p. 2057-99.

[3] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.; US EPA. Integrated Science Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA/600/R-08/139F. December 2009. Includes errata sheet created on 2/10/2010, 2009.; US EPA. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. Report No.: EPA 452/R-11-003. April, 2011.; US EPA. Provisional assessment of recent studies on health effects of particulate matter exposure. Research Triangle Park, NC. National Center for Environmental Assessment-RTP Division; Office of Research and Development; US EPA, Report No.: EPA/600/R-12/056F. December, 2012.; US EPA. Quantitative Health Risk Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA-452/R-10-005. June, 2010.

disease and lung cancer, reduced lung development and the development of chronic respiratory diseases (e.g., asthma) in children, aggravated asthma, decreased lung function, and increased respiratory symptoms such as irritation of the airways, coughing, or difficulty breathing.[4]

B. Scientific Literature and Historical Precedence Establish that Exceedance of NAAQS Is Not Required for $PM_{2.5}$ to be Considered Potentially Harmful—There is No Identifiable Threshold Concentration Below Which Adverse Health Effects Would Not Occur.

5. Dr. Maier's declaration states that "In setting the NAAQS, the EPA must consider 'the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare that [*sic*] may be expected from the presence of [$PM_{2.5}$] in the ambient

[4] American Lung Association. State of the Air 2017. Chicago, IL. 2017., p.35; Bell, M.L., *et al.* Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. Am J Epidemiol. 168(11):1301-10, 2008.; Delfino, R.J., *et al.* Asthma morbidity and ambient air pollution: effect modification by residential traffic-related air pollution. Epidemiology. 25(1):48-57, 2014.; Dominici, F., *et al.* Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 295(10):1127-34, 2006.; Krewski, D., *et al.* Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution and mortality. Cambridge, MA. Contract No.: A Special Report of the Institute's Particle Epidemiology Reanalysis Project. July, 2000.; Krewski, D., *et al.* Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Boston, MA. Contract No.: HEI Research Report 140. May, 2009.; Laden, F., *et al.* Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six cities study. Am J Respir Crit Care Med. 173(6):667-72, 2006.; Moolgavkar, S.H. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Metzger, K.B., *et al.* Ambient air pollution and cardiovascular emergency department visits. Epidemiology. 15(1):46-56, 2004.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 117(3):475-80, 2009.; Pope, C.A., III, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 287(9):1132-41, 2002.; Pope, C.A., III and Dockery, D.W. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc. 56(6):709-42, 2006.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 124(1):46-52, 2016.; US EPA. Particulate Matter (PM) Pollution: Health and Environmental Effects of Particulate Matter (PM). Last updated July 1, 2016. Available from: https://www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm. Accessed: 9/21/2017. ; Zanobetti, A. and Schwartz, J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 117(6):898-903, 2009.

air, in varying quantities.' 42 U.S.C. § 7408(a)(2). The standard is set at a level which is protective of 'the health of 'sensitive' populations such as asthmatics, children, and the elderly,' [ ] with an adequate margin of safety for protecting those sensitive individuals built into the EPA standard. 42 U.S.C. § 7409(b)(1)." (Maier Decl., ¶ 5.)

6. Dr. Maier's declaration fails to acknowledge that scientific studies have not shown a "threshold" concentration (the exposure concentration at which an adverse effect is first observed) for adverse health effects associated with ambient exposure to $PM_{2.5}$. Without a threshold for response, numerous scientific studies have indicated $PM_{2.5}$ is potentially harmful below the current NAAQS.[5]

7. The failure of scientific studies to identify a threshold for $PM_{2.5}$ exposure concentrations and associated adverse human health effects has been acknowledged by the World Health Organization, the US EPA, the Clean Air Scientific Advisory Committee (CASAC), the Advisory Council on Clean Air Compliance Analysis, the American Heart Association, and numerous epidemiology studies.[6]

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[5] Fann, N., *et al.* Estimated Changes in Life Expectancy and Adult Mortality Resulting from Declining PM2.5 Exposures in the Contiguous United States: 1980–2010. Environ Health Perspect. (9), 2017.; Lee, M., *et al.* Acute effect of fine particulate matter on mortality in three Southeastern states from 2007-2011. J Expo Sci Environ Epidemiol. 26(2):173-9, 2016.; Makar, M., *et al.* Estimating the Causal Effect of Low Levels of Fine Particulate Matter on Hospitalization. Epidemiology. 28(5):627-34, 2017.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 124(1):46-52, 2016.

[6] Bell, M.L., *et al.* Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. Am J Epidemiol. 168(11):1301-10, 2008.; Delfino, R.J., *et al.* Asthma morbidity and ambient air pollution: effect modification by residential traffic-related air pollution. Epidemiology. 25(1):48-57, 2014.; Dominici, F., *et al.* Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 295(10):1127-34, 2006.; Krewski, D., *et al.* Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution and mortality. Cambridge, MA. Contract No.: A Special Report of the Institute's Particle Epidemiology Reanalysis Project. July, 2000.; Krewski, D., *et al.* Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Boston, MA. Contract No.: HEI Research Report 140. May, 2009.; Laden, F., *et al.* Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six cities study. Am J Respir Crit Care Med. 173(6):667-72, 2006.; Moolgavkar, S.H. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series

8. US EPA recognizes the NAAQS is not a threshold below which no $PM_{2.5}$ effects would be expected in exposed communities. In setting the current NAAQS, the US EPA calculated attributable disease incidence for lesser exposure concentrations (alternate annual averaged $PM_{2.5}$ concentrations of 10, 12, 13, and 14 $\mu g/m^3$ combined with different 24-hr standards). Selection of evaluated annual values was based on the available epidemiological evidence (i.e., the current exposure-dose-response continuum characterized in the scientific literature).[7] Evaluated alternate annual scenarios were combined with different 24-hour standard scenarios to generate risk metrics (i.e., annual incidence of endpoints due to exposure). In using the above approach to setting the standard, the US EPA recognized that, compared to ambient $PM_{2.5}$ concentrations from 2005, not only would the annual incidence of adverse health effects (mortality, hospital admissions) be reduced with the eventually promulgated 2012 $PM_{2.5}$ standards (35 and 12 $\mu g/m^3$ for 24-hr and annual levels, respectively), but also that the annual incidence of adverse health effects would be further reduced at NAAQS below the 2012 levels.[8] **Therefore, the US EPA recognizes the NAAQS is not a threshold for $PM_{2.5}$ effects in exposed communities.**

Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Metzger, K.B., *et al.* Ambient air pollution and cardiovascular emergency department visits. Epidemiology. 15(1):46-56, 2004.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 117(3):475-80, 2009.; Pope, C.A., III, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 287(9):1132-41, 2002.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 124(1):46-52, 2016.; Zanobetti, A. and Schwartz, J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 117(6):898-903, 2009.; H. Nadia Moore, Ph.D., DABT, ERT Rebuttal Expert Report in the matter of Oakland Bulk & Oversized Terminal, LLC v. City of Oakland, Sierra Club and San Francisco Baykeeper. Case No. 3.16-cv-07014-VC.

[7] US EPA. Quantitative Health Risk Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA-452/R-10-005. June, 2010.

[8] US EPA. Quantitative Health Risk Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA-452/R-10-005. June, 2010., Appendix E and J.

C. NAAQS Values for Criteria Pollutants, Set By the United States Environmental Protection Agency, Are Dynamic and Evolve with Scientific Knowledge Regarding Adverse Health Effects—Harms to Health from Increased Criteria Pollutants are Possible Below NAAQS Values.

9. Dr. Maier's declaration states that National Ambient Air Quality Standards ("NAAQS"), including NAAQS for $PM_{2.5}$, "quantify the levels at which pollutants 'may reasonably be anticipated to endanger public health or welfare.' 42 U.S.C. § 7408(a)(1)(A)." (Maier Decl., ¶ 3.) He further states that "NAAQS represents the 'ambient air quality standards the attainment and maintenance of which in the judgment of the [EPA], based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.' 42 U.S.C. § 7409(b)(1) (emphasis omitted)." (Maier Decl., ¶ 4.)

10. Dr. Maier's declaration correctly references relevant statutory provisions but does not address the dynamic nature and historical evolution of the NAAQS. Specifically, **NAAQS** are established as **policy decisions** whereby the US EPA Administrator uses current scientific datasets to establish and set, to the best of her or his judgment, **levels <u>neither more nor less stringent</u> than necessary to provide the requisite degree of public health protection**, including the health of at-risk populations, with an adequate margin of safety.[9]

11. Ambient air quality in the United States has improved following promulgation of the NAAQS[10] and the US EPA has acknowledged the tendency for implemented NAAQS to lower the overall air quality distribution (concentration) for the population.[11] In other words, promulgating an NAAQS results in lowering the average population exposure concentration, usually below the NAAQS concentration.

12. NAAQS for PM-attributed health effects are based on human epidemiology studies, whose results (1) have not identified a threshold concentration below which adverse

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[9] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.

[10] EPA, U. Air Quality - National Summary. Last updated July 26, 2017. Available from: https://www.epa.gov/air-trends/air-quality-national-summary. Accessed: 11/29/2017.

[11] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.

health effects would not occur[12] and (2) are fundamentally linked with the ambient concentrations experienced by the study population.[13] The overall improvement of ambient air concentrations following implementation of NAAQS resulted in the ability of subsequent epidemiology studies to evaluate populations exposed to lesser average concentrations. These subsequent epidemiology studies and their observations of associations with adverse health effects at lesser ambient $PM_{2.5}$ concentrations formed the scientific basis for the reduction in the annual $PM_{2.5}$ NAAQS from 15.0 $\mu g/m^3$ (set in 1997) to 12.0 $\mu g/m^3$ (set in 2012) because, for example:

> "…even more so than in the last review, the **evidence indicates a 'significant public health risk' to children from long-term $PM_{2.5}$ exposures at concentrations below the level of the current annual standard.**" [emphasis added][14]

13. In addition, the US EPA's Final Rule indicated the collective conclusion of its Policy Assessment regarding PM NAAQS was:

> "…that currently available evidence provided support for **associations between long-term $PM_{2.5}$ exposure and mortality and morbidity effects that extend to distributions of $PM_{2.5}$ concentrations that are lower than those that had previously been associated with such effects**, with aggregate long-term mean $PM_{2.5}$ concentrations extending to well below the level of the current annual standard." [emphasis added][15]

14. Put another way, the best way to study the potential impact of $PM_{2.5}$ exposures on mortality and morbidity effects is to evaluate potential effects "after" a reduction in ambient concentrations. Such an opportunity occurs upon establishing (or lowering) the NAAQS, because the overall national air quality distribution (concentration) generally decreases in order to meet a new (or lesser) NAAQS.[16] Thus, studies "before" provide the scientific basis for the established

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[12] H. Nadia Moore, Ph.D., DABT, ERT Rebuttal Expert Report in the matter of Oakland Bulk & Oversized Terminal, LLC v. City of Oakland, Sierra Club and San Francisco Baykeeper. Case No. 3.16-cv-07014-VC, p. 9-13. [Excerpt attached hereto as Exhibit 2.]

[13] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.

[14] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013., p.3152.

[15] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013., p.3107; US EPA. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. Report No.: EPA 452/R-11-003. April, 2011.

[16] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40

NAAQS while studies "after" provide scientific evaluations of populations exposed to lesser $PM_{2.5}$ concentrations (i.e., population exposure concentrations not previously available in earlier assessments). Such "after" circumstances have not yet determined the threshold below which no effects occur, but did form the basis for the 2012 reduction in the annual $PM_{2.5}$ NAAQS level.[17]

15. To summarize, the NAAQS are established as policy judgments, based on the current understanding of the available scientific literature, as the maximum permissible ambient air concentration for each criteria pollutant that will protect the health of any [sensitive] group of the population.[14] As population exposures to PM have declined over time, subsequent studies of lesser-exposed populations continued to demonstrate effects at lesser ambient concentrations, driving NAAQS levels to lesser concentrations. **Therefore, the NAAQS reflects the most current scientific knowledge available to protect human health and should not be regarded as a "bright line" (or threshold) between an effect and a no effect level.**

D. ESA's Conclusions Were Based on Nonattainment Classification and Cumulative Effects.

16. Finally, the ESA Report prepared for the City of Oakland in connection with the administrative record concerning the subject Ordinance contained a discussion that included current values (levels) established for the Federal (NAAQS) and State Ambient Air Quality Standards for $PM_{2.5}$ and other criteria pollutants. ESA explained that the US EPA classifications of "attainment" or "nonattainment" are based on whether or not NAAQS levels have been achieved. The ESA Report summarized that the Bay Area was then currently classified as "nonattainment" for the 24-hr $PM_{2.5}$ NAAQS and that the West Oakland community had observed exceedances in both the 24-hr and annual average NAAQS for $PM_{2.5}$."[18] In evaluating

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CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013., p.3163.

[17] US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.

[18] ESA. Report on the Health and/or Safety Impacts Associated with the Transport, Storage, and/or Handling of Coal and/or Coke in Oakland, including at the Proposed Oakland Bulk and Oversized Terminal in the West Gateway Area of the former Oakland Army Base. Prepared for the City of Oakland. June 23, 2016 ("ESA Report"). (Myre Decl., Ex. 45, pp. 4-2, 4-3, 4-11).

the proposed coal-handling and operations, ESA estimated the $PM_{2.5}$ emissions into the ambient air that would occur under OBOT's proposed conditions, reporting the amount of $PM_{2.5}$ emissions from fugitive coal dust into the ambient air "from rail transport, staging/spur travel, unloading, storage, transfer and ship loading of coal at OBOT" in units of tons/yr.[19] The ESA conclusion regarding health impacts appears to be based on the propensity for fugitive coal dust to add to the then-current "nonattainment" conditions of West Oakland. Specifically: "Based upon the total emissions estimates for fugitive coal dust for all activities associated with OBOT and the re-entrainment of accumulated fugitive coal dust, we conclude that these cumulative contributions of particulates to local levels of TSP, PM_{10}, and $PM_{2.5}$ would further degrade existing air quality. This cumulative contribution would be likely to cause additional exceedances of ambient air quality standards for PM_{10} and particularly $PM_{2.5}$, at the air monitoring station in West Oakland. Since the ambient air quality would not improve, and the standards would likely continue to be exceeded, this degraded local air quality would continue to impact the health of the adjacent neighbors."[20]

I declare under penalty of perjury under the laws of the United States that the foregoing is true and correct. Executed this 5th day of December, 2017, at Redmond, Washington.

/s/ H. Nadia Moore, Ph.D., DABT, ERT
H. NADIA MOORE, PH.D., DABT, ERT

[19] ESA Report (Myre Decl., Ex. 45, p. 5-17).

[20] ESA Report (Myre Decl., Ex. 45, p. 5-17).

ATTESTATION

I, Kevin D. Siegel, am the ECF user whose ID and password are being used to file this "Declaration of H. Nadia Moore, Ph.D., Dabt, Ert in Support of Defendant City of Oakland's Motion for Summary Judgment, Or In The Alternative Partial Summary Judgment, and Opposition to Plaintiff's Motion for Summary Judgment." Pursuant to Civil Local Rule 5-1(i)(3), I hereby attest that H. Nadia Moore has concurred in the filing of this document.

DATED: December 5, 2017

/s/ Kevin D. Siegel
Kevin D. Siegel

EXHIBIT 1

VERITOX®
REDMOND, WA • HILTON HEAD, SC

Hope (Nadia) Moore, PhD, DABT, ERT

Education

Ph.D., Toxicology, University of Washington, Seattle WA, 2008

B.S., Chemistry, Pacific Lutheran University, Tacoma, WA, 1992

Certifications

Diplomate of the American Board of Toxicology, 2012-present

Registered Toxicologist (United Kingdom and EUROTOX registries), 2015-present

NIOSH Spirometry Testing Certification, 2015-present

Project Management Professional, 2011-2014

Professional Affiliations and Associated Appointments

American Board of Toxicology

Society of Toxicology (SOT), Full member
- Inhalation and Respiratory Specialty Section member
- Nanotoxicology Specialty Section member
- Women in Toxicology Interest Group member
  - WIT Councilor (2016-2018 term)
- Pacific Northwest Association of Toxicologists (PANWAT) member
  - PANWAT Councilor (2014-2016 term)
  - PANWAT Vice President Elect (2016-2017 term)
  - PANWAT Vice President (2017-2018 term)

American College of Toxicology (ACT), Full member

British Toxicology Society (BTS), Member

American College of Occupational and Environmental Medicine (ACOEM), Associate member

American Association for the Advancement of Science (AAAS), Member

American Conference of Governmental Industrial Hygienists (ACGIH), Voting member

American Chemical Society (ACS), Member

American Industrial Hygiene Association (AIHA), Full member

Society for Experimental Biology and Medicine (SEBM), Associate member

Hope (Nadia) Moore, PhD, DABT, ERT

Other Professional Activities

Practicing Scientist Member, Institutional Animal Care and Use Committee (IACUC) Pacific Northwest National Laboratory, Sequim Laboratory, and the Columbus-based Toxicology Laboratory (ToxNW), 2011 - 2013

Invited lecturer, Fundamentals of Toxicology Graduate Course ENVH514, University of Washington (Lung: Structure, Function, Absorption, & Inhalation)

Invited reviewer, Food and Chemical Toxicology (Elsevier Sciences), Human and Experimental Toxicology (Sage Journals), NeuroToxicology (Elsevier Sciences), Toxicology Letters (Elsevier Sciences), and Toxicological Sciences (Oxford Journals)

Experience

2013 - Present	**Veritox®, Inc.** *Senior Toxicologist*	**Redmond, Washington**
2008-2013	**Battelle Toxicology Northwest** *Pharmacologist / Safety Toxicologist*	**Richland, Washington**
2003-2008	**University of Washington** *Toxicology Doctoral Student / Teaching Assistant / Research Assistant*	**Seattle, Washington**
2001-2003	**Battelle Pacific NW National Laboratory** *Senior Research Scientist*	**Richland, Washington**
2000-2001	**Battelle Toxicology Northwest** *Principal Research Scientist*	**Richland, Washington**
1992-2000	**Battelle Toxicology Northwest** *Research Scientist / Technical Specialist /Technician*	**Richland, Washington**

Professional Honors

National Toxicology Program Toxicology Discipline Leader Battelle Toxicology Northwest, Richland, WA (2011-2013)

Outstanding Performance Award in recognition of Outstanding Efforts as Study Director. Battelle Toxicology Northwest, Richland, WA (2009)

Pre-Doctoral Fellow National Institute of Environmental Health Sciences Environmental Pathology and Toxicology Training Grant.
University of Washington, Seattle, WA (2005-2008)

Outstanding Student Poster Award Recipient from the PANWAT (Pacific Northwest Association of Toxicologists, Regional Chapter of the Society of Toxicology) Annual Meeting (2007)

Hope (Nadia) Moore, PhD, DABT, ERT

Student Merit Meeting Award Recipient from the Research Society on Alcoholism, 30th Annual Meeting of the Research Society on Alcoholism, Chicago, IL (2007)

Selected Publications

TJ Mast, F Adeshina, N Moore, H Choudhury, A Protzel, and A Mahfouz. 2002. Identification of common toxic effects with common mechanisms of toxicity for pesticides selected from the drinking water contaminant list (CCL). *The Toxicologist,* Supplement to Toxicological Sciences, 66(1):494.

F Adeshina, T Mast, N Moore, A Mahouz, A Protzel, and H Choudhury. 2003. Identifying triazine herbicides on EPA drinking water contaminant candidate list (CCL) for common mechanism of toxicity and cumulative risk assessment. *The Toxicologist,* Supplement to Toxicological Sciences, 72(1):436.

N Moore, M Guizzetti, B Gallis, S Shaffer, DR Goodlett, and LG Costa. 2006. Use of proteomic approaches for the identification of changes in astrocyte secretion following ethanol exposure. *The Toxicologist,* Supplement to Toxicological Sciences, 90(1):1437.

M Guizzetti, G Giordano, N Moore, and LG Costa. 2008. Ethanol inhibits hippocampal neuron differentiation induced by carbachol-treated astrocytes. *The Toxicologist,* Supplement to Toxicological Sciences, 102(1):1963.

M Guizzetti, NH Moore, G Giordano, and LG Costa. 2008. Modulation of neuritogenesis by astrocyte muscarinic receptors. *J. Biol. Chem.* 283(46): 31884-31897.

N Moore, M Guizzetti, G Giordano, and LG Costa. 2008. Ethanol inhibits muscarinic receptor-induced release by astrocytes of extracellular proteins involved in neuronal development. *The Toxicologist,* Supplement to Toxicological Sciences, 102(1):1964.

NH Moore, LG Costa, SA Shaffer, DR Goodlett, and M Guizzetti. 2009. Shotgun proteomics implicates extracellular matrix proteins and protease systems in neuronal development induced by astrocyte cholinergic stimulation. *J. Neurochem.* 108(4):891-908.

M Guizzetti, NH Moore, G Giordano, KL VanDeMark, and LG Costa. 2010. Ethanol inhibits neuritogenesis induced by astrocyte muscarinic receptors. *Glia.* 58(12):1395-406.

M Guizzetti, NH Moore, KL VanDeMark, G Giordano, and LG Costa. 2011. Muscarinic receptor-activated signal transduction pathways involved in the neuritogenic effect of astrocytes in hippocampal neurons. *Eur. J. Pharmacol.* 659(2-3):102-7.

CY Chan, LJ Swenson, J Hobden, N Moore, and BJ Kelman. 2014. Risk from exposure to triorthocresyl phosphate (TOCP) in aircraft cabins and flight decks. *The Toxicologist,* Supplement to Toxicological Sciences, 138(1):2247.

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Selected Continuing Education

Analytical Validation for the Pharmaceutical Industry, American Association of Pharmaceutical Scientists (AAPS). AAPS Workshop on Current Issues, Arlington, VA, 1998.

Bioanalytical Methods Validation – A Revisit with a Decade of Progress. Co-sponsored by AAPS and FDA, Arlington, VA, 2000.

GLP Essentials for Technical Staff. Debi Garvin, Instructor, West Coast Quality Training Institute, Richland, WA, 2001.

A Practical Approach to Blood and Lymphoid Tissue (BLT) in Toxicology Assessments. JCL Schuh and L Lanning, Chairpersons. Society of Toxicology Continuing Education Course, Nashville, TN, 2002.

Good Laboratory Practices for Study Directors and Monitors. D Garvin, Instructor and Director, West Coast Quality Training Institute, Hood River, OR, 2008.

Introduction to Good Laboratory Practice Regulations. D Garvin, Instructor and Director, West Coast Quality Training Institute, Hood River, OR, 2008.

Primer in Pathology: Interpreting and Integrating Nonclinical Study Results. Continuing Education Course. Pacific Northwest Chapter of the Society of Toxicology, Pacific Northwest Association of Toxicologists Annual Meeting, Seattle, WA, 2009.

Stress as a Confounding Factor in Toxicology Studies. K Sprugel and N Everds, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2009.

Immunology for Toxicologists. R Pieters and I Kimber, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2009.

Integrative Toxicity Test Methods to Improve Hazard Identification. Society of Toxicology Pacific Northwest Chapter, Pacific Northwest Association of Toxicologists Annual Meeting, Corvallis, OR, 2010.

Segment-Specific Renal Pathology for the Non-Pathologist. D Hoivik and SG Emeigh Hart, Chairpersons. Society of Toxicology Continuing Education Course, Salt Lake City, UT, 2010.

Evaluating Toxicity of Engineered Nanomaterials: Issues with Conventional Toxicology Approaches. SS Nadadur and FA Witzmann, Chairpersons. Society of Toxicology Continuing Education Course, Washington DC, 2011.

Current Nonclinical Strategies and Methods for Evaluating Drug-Induced Cardiovascular Toxicity. H Wang and DJ. Murphy, Chairpersons. Society of Toxicology Continuing Education Course, Washington DC, 2011.

Northwest Association for Biomedical Research IACUC Education Conference, Seattle, WA, 2011.

Art and Science of Research Translation in Toxicology. Society of Toxicology Pacific Northwest Chapter, Pacific Northwest Association of Toxicologists Annual Meeting. North Bonneville, WA, 2011.

Mid America Toxicology Course, CD Klaassen, Course Director, Kansas City, Missouri, April 2012.

The What, When, and How of Nonclinical Support for an IND Submission. P Nugent and D Colagiovanni, Chairpersons. Society of Toxicology Continuing Education Course, San Antonio, TX, 2013.

Understanding Toxic Neuropathy in Drug Development: Both Clinical and Nonclinical Perspectives. MJ Kallman and J Benitez, Chairpersons. Society of Toxicology Continuing Education Course, San Antonio, TX, 2013.

Innovations in Methodologies for Inhalation Exposures and Interpretations of In Vivo Toxicity, Urmila Kodavanti and Juergen Pauluhn, Chairpersons. Society of Toxicology Continuing Education Course, Phoenix, AZ, 2014.

Methodologies in Human Health Risk Assessment, Qiyu (Jay) Zhao and M.E. (Bette) Meek, Chairpersons. Society of Toxicology Continuing Education Course, Phoenix, AZ, 2014.

Advances in Safety Assessment of Medical Devices, Niranjan S Goud and Ron Brown, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

Hope (Nadia) Moore, PhD, DABT, ERT

New Horizons in Chemical Carcinogenesis: Advances in Mode of Action and Mechanism of Cancer Pathogenesis, James E Klaunig and Udayan M Apte, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

The New World of Cancer Immunotherapy: Challenges in Bench to Bedside Translation, Rodney Prell and Rafael A Ponce, Chairpersons. Society of Toxicology Continuing Education Course, San Diego, CA, 2015.

OECD GLP and Documentation Training Course, Robin Guy and Dave Hobson, Instructors. Robin Guy Consulting, Morristown, NJ, Aug. 31 – Sept. 1, 2015.

Nanomaterials, Chemical Exposures and Control Banding: What Does It Mean for Workplace Safety? University of Washington Continuing Education Programs, hosted by the Pacific Northwest Section – American Industrial Hygiene Association. Oct. 14, 2015

NIOSH-Approved 2-Day Initial Spirometry Training Course, Martha Horike-Pyne, Instructor. University of Washington Continuing Education Programs, Seattle WA. Nov. 7-8, 2015.

Advancing the Detection, Imaging, and Pitfalls in Monitoring Oxidative Stress in Health and Disease. Maria B. Kadiiska and Ronald P. Mason, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

Basic Principles and Practices for Applying Epigenetics in Mechanistic Toxicology. Shaun D. McCullough and Ronald N. Hines, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

Human Health Risk Assessment: A Case Study Application of Principles. John C. Lipscomb and M.E. (Bette) Meek, Chairpersons. Society of Toxicology Continuing Education Course, New Orleans, LA, 2016.

Adding Up Chemicals: Component-Based Risk Assessment of Chemical Mixtures. Jane Ellen Simmons and Richard C. Hertzberg, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2017.

Extrapolation in the Airways: Strategies to Incorporate *In Vivo* and *In Vitro* Data to Better Protect Human Health. Marie C. Fortin and Madhuri Singal, Chairpersons. Society of Toxicology Continuing Education Course, Baltimore, MD, 2017.

EXHIBIT 2

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October 16, 2017

Gregory Aker, Esq.
Burke Williams & Sorensen LLP
1901 Harrison Street #900
Oakland, CA 94612

RE: Supplemental Expert Report of H. Nadia Moore, Ph.D., DABT, ERT in the matter of Oakland Bulk & Oversized Terminal, LLC v. City of Oakland, Sierra Club and San Francisco Baykeeper. Case No. 3.16-cv-07014-VC

Dear Mr. Aker:

I was asked to supplement my Expert Report dated October 6, 2017, with the revised modeling results provided by Dr. Gray. This entailed revision of my original estimates of airborne $PM_{2.5}$ pollution levels that supported my opinions regarding the effect of coal transport, storage, and handling to ambient $PM_{2.5}$ pollution levels for the West Oakland community.

Summary of primary opinions

Inclusion of Dr. Gray's revised dispersion modeling results for coal transport, storage, and handling of coal increased the expected ambient $PM_{2.5}$ pollution levels in the West Oakland community, but did not change my conclusion that coal operations will pose an increased risk of adverse health effects to the West Oakland community. Specifically:

- **Supplemental Opinion 1: Revised modeling results provide added support that transport, storage, and handling of coal through the City and Port of Oakland poses an increased risk of adverse health effects to West Oakland residents, including premature mortality, increased hospital admissions and emergency department visits, and development of chronic respiratory disease** (page 4).
    - Modeled coal-associated $PM_{2.5}$ concentrations cause West Oakland's ambient air quality to exceed National (NAAQS) and State (CAAQS) air quality standards.

Conceptually, a threshold for $PM_{2.5}$ effects exists; scientific literature has not yet identified it

Dr. Maier indicates that thresholds associated with "safe" exposure concentrations are "expected to exist,"[13] but he fails to acknowledge the vast set of scientific literature that has agreed to date **no threshold** has been established for adverse effects of $PM_{2.5}$ exposure. His critique of others' opinions that exposure to $PM_{2.5}$ at levels below the NAAQS are not safe[14] is contrary to the breadth of scientific literature, regulatory organizations, and expert panels that have failed to identify a threshold for effects.

Specifically, numerous epidemiology studies have characterized dose-response relationships between $PM_{2.5}$ exposure and adverse human health effects but have failed to identify the threshold for the effects.[15] For example, a meta-analysis (i.e., analysis of data

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Epidemiology. 15(1):46-56, 2004.; Moolgavkar, S.H. Air pollution and daily deaths and hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 117(3):475-80, 2009.;Pope, C.A., III, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 287(9):1132-41, 2002.; Pope, C.A., III and Dockery, D.W. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc. 56(6):709-42, 2006.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 124(1):46-52, 2016.; Metzger, K.B., *et al.* Ambient air pollution and cardiovascular emergency department visits. Epidemiology. 15(1):46-56, 2004.; US EPA. National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.; US EPA. Integrated Science Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA/600/R-08/139F. December 2009. Includes errata sheet created on 2/10/2010, 2009.; US EPA. Policy Assessment for the Review of the Particulate Matter National Ambient Air Quality Standards. Report No.: EPA 452/R-11-003. April, 2011.; US EPA. Provisional assessment of recent studies on health effects of particulate matter exposure. Research Triangle Park, NC. National Center for Environmental Assessment-RTP Division; Office of Research and Development; US EPA, Report No.: EPA/600/R-12/056F. December, 2012.; US EPA. Quantitative Health Risk Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA-452/R-10-005. June, 2010.; US EPA. Particulate Matter (PM) Pollution: Health and Environmental Effects of Particulate Matter (PM). Last updated July 1, 2016. Available from: https://www.epa.gov/pm-pollution/health-and-environmental-effects-particulate-matter-pm. Accessed: 9/21/2017. ; Zanobetti, A. and Schwartz, J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 117(6):898-903, 2009.

[13] Andrew Maier. Expert Report of Andrew Maier. Oakland Bulk & Oversized Terminal, LLC v. City of Oakland. Civil Action No. 16-07014(VC), October 6, 2017., p.9.

[14] Andrew Maier. Expert Report of Andrew Maier. Oakland Bulk & Oversized Terminal, LLC v. City of Oakland. Civil Action No. 16-07014(VC), October 6, 2017., p.12.

[15] Bell, M.L., *et al.* Seasonal and regional short-term effects of fine particles on hospital admissions in 202 US counties, 1999-2005. Am J Epidemiol. 168(11):1301-10, 2008.; Delfino, R.J., *et al.* Asthma morbidity and ambient air pollution: effect modification by residential traffic-related air pollution. Epidemiology. 25(1):48-57, 2014.; Dominici, F., *et al.* Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 295(10):1127-34, 2006.; Krewski, D., *et al.* Reanalysis of the Harvard six cities study and the American Cancer Society study of particulate air pollution and mortality. Cambridge, MA. Contract No.: A Special Report of the Institute's Particle Epidemiology Reanalysis Project. July, 2000.; Krewski, D., *et al.* Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality. Boston, MA. Contract No.: HEI Research Report 140. May, 2009.; Laden, F., *et al.* Reduction in fine particulate air pollution and mortality: extended follow-up of the Harvard six cities study. Am J Respir Crit Care Med. 173(6):667-72, 2006.; Moolgavkar, S.H. Air pollution and daily deaths and

from multiple studies) indicated that the best empirical evidence suggests that, across the range of $PM_{2.5}$ pollution observed in most recent studies, that the

> *"Estimated concentration-response functions are near linear, with* ***no evidence of safe threshold levels*** [emphasis added].[16]

Regulatory organizations agree studies have failed to identify a threshold between $PM_{2.5}$ exposure and adverse human health effects:

- 2006: The World Health Organization (WHO) stated:

  > *"Current scientific evidence indicates that guidelines cannot be proposed that will lead to complete protection against adverse health effects of PM, as* ***thresholds have not been identified****. Rather, the standard-setting process needs to achieve the lowest concentrations possible in the context of local constraints, capabilities and public health priorities"* [emphasis added][17]

- 2009: US EPA Integrated Science Assessment:

  > *"Although multiple studies have previously examined the PM-mortality concentration-response relationship and whether a threshold exists, more complex statistical analyses continue to be developed to analyze this association. ... Overall, the studies evaluated* ***further support the use of a no-threshold log-linear model****, but additional issues such as the influence of heterogeneity in estimates between cities, and the effect of seasonal and regional differences in*

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hospital admissions in Los Angeles and Cook Counties. In: HEI Special Report Revised Analyses of Time-Series Studies of Air Pollution and Health Revised Analyses of the National Morbidity, Mortality, and Air Pollution Study, Part II Revised Analyses of Selected Time-Series Studies. Boston, MA: Health Effects Institute; 2003. p. 183-98.; Metzger, K.B., *et al.* Ambient air pollution and cardiovascular emergency department visits. Epidemiology. 15(1):46-56, 2004.; Ostro, B., *et al.* The effects of fine particle components on respiratory hospital admissions in children. Environ Health Perspect. 117(3):475-80, 2009.;Pope, C.A., III, *et al.* Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. JAMA. 287(9):1132-41, 2002.; Shi, L., *et al.* Low-Concentration PM2.5 and Mortality: Estimating Acute and Chronic Effects in a Population-Based Study. Environ Health Perspect. 124(1):46-52, 2016.; Zanobetti, A. and Schwartz, J. The effect of fine and coarse particulate air pollution on mortality: a national analysis. Environ Health Perspect. 117(6):898-903, 2009.

[16] Pope, C.A., III and Dockery, D.W. Health effects of fine particulate air pollution: lines that connect. J Air Waste Manag Assoc. 56(6):709-42, 2006.

[17] Samet, J.M., *et al.* Particulate Matter. In: Air Quality Guidelines Global Update 2005: Particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Germany: World Health Organization; Druckpartner Moser; 2006. p. 217-91.

PM on the concentration-response relationship still require further investigation." [emphasis added][18]

- 2013: In the Final Rule for NAAQS $PM_{2.5}$ levels, the US EPA Administrator

  "[recognized] that the health effects may occur over the full range of concentrations observed in the epidemiological studies of both long-term and short-term exposures, ***since no discernible population-level threshold for any such effects can be identified based on the currently available evidence****"*[emphasis added][19]

Independent expert scientific panels agree studies have failed to identify a threshold between $PM_{2.5}$ exposure and adverse human health effects:

- The Clean Air Scientific Advisory Committee (CASAC) provides independent scientific advice to the EPA Administrator on the technical basis for the US EPA's NAAQS.[20] The independent CASAC review of Second External Review Draft of the PM NAAQS Policy Assessment stated:

  "The conclusions [regarding consideration of alternate/lower standards] are reasonable in relation to the criteria established by the Clean Air Act (CAA)... choices within these options will need to be based on the Administrator's interpretation of the CAA's requirement for an adequate margin-of-safety. In other words, ***in the absence of thresholds in the dose-response relationships for the health outcomes of concern****, [the Administrator will need to decide] how*

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[18] US EPA. Integrated Science Assessment for Particulate Matter. Research Triangle Park, NC. Report No.: EPA/600/R-08/139F. December 2009. Includes errata sheet created on 2/10/2010, 2009.

[19] US EPA, National ambient air quality standards for particulate matter. Final Rule. 40 CFR Parts 50, 51, 52, 53, and 58. Federal Register. 78(10):3086-287, 2013.

[20] CASAC is required by Section 109 of the Clean Air Act (CAA) to review NAAQS criteria, standards, and serve as a scientific/technical advisory committee to the US EPA. CASAC is comprised of 7 members including at least one member of the National Academy of Sciences, one physician, and one person representing State air pollution control agencies. All members have demonstrated high levels of competence, knowledge, and expertise in scientific/.technical fields relevant to air pollution and air quality issues.US EPA. EPA Clean Air Scientific Advisory Committee (CASAC). Last updated January 21, 2016. Available from: https://yosemite.epa.gov/sab/sabpeople nsf/WebCommittees/CASAC. Accessed: 10/12/2017. ; US EPA. United States Environmental Protection Agency Charter. Clean Air Scientific Advisory Committee. Agency Approved May 23, 2017, Filed with Congress June 5, 2017. 2017.

much public health impact resulting from exposure to ambient air $PM_{2.5}$ is acceptable under the CAA"[21]

- The Advisory Council on Clean Air Compliance Analysis (Council), active from 1991 – 2014, provided advice, information, and recommendations to the US EPA regarding the costs and benefits of the CAA.[22] The Council established a Health Effects Subcommittee (HES) to review and provide guidance to the US EPA on the draft human health effect estimates in the second Section 812 Prospective Analysis benefits report as well as the human health components for the draft stand-alone uncertainty analysis report. They stated:

  *"The **HES fully supports EPA's decision to use a no-threshold model** to estimate mortality reductions. This decision is supported by the data, which are quite consistent in showing effects down to the lowest measured levels. Analyses of cohorts using data from more recent years, during which time PM concentrations have fallen, continue to report strong associations with mortality. Therefore, there is no evidence to support a truncation of the CRF [concentration-response function]"* [emphasis added][23]

Independent expert scientific organizations agree studies have failed to identify a threshold between $PM_{2.5}$ exposure and adverse human health effects:

- The American Heart Association evaluated the scientific literature for PM air pollution and cardiovascular disease and concluded:

  *"The $PM_{2.5}$ concentration– cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 $\mu g/m^3$ (the 2006 annual NAAQS level) **without a discernable "safe" threshold**. ...*

  *... Because **the evidence reviewed supports that there is no safe threshold**, it appears that public health benefits would accrue from lowering $PM_{2.5}$*

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[21] Samet, J. Letter from Dr. Jonathan M. Samet, Chair, Clean Air Scientific Advisory Committee to the Honorable Lisa P. Jackson, Administrator, US EPA. CASAC Review of Policy Assessment for the Review of the PM NAAQS – Second External Review Draft (June 2010). Clean Air Scientific Advisory Committee (CASAC). September 10, 2010.

[22] US EPA. EPA Advisory Council on Clean Air Compliance Analysis. Last updated January 8, 2016. Available from: https://yosemite.epa.gov/sab/sabpeople nsf/WebCommittees/COUNCIL/. Accessed: 10/12/2017.

[23] Hammitt, J.K. and Bailar, J. Letter from Dr. James K. Hammitt, Chair, Advisory Council on Clean Air Compliance Analysis and Dr. John Bailar, Chair Health Effects Subcommittee to the Honorable Lisa P. Jackson, Administrator, US EPA. Review of EPA's DRAFT Health Benefits of the Second Section 812 Prospective Study of the Clean Air Act. June 16, 2010.

> *concentrations even below present-day annual (15 µg/m^3) and 24-hour (35 µg/m^3) NAAQS, if feasible, to optimally protect the most susceptible populations"* [emphasis added][24]

NAAQS are not based on absolute "thresholds" for adverse effects

Dr. Maier's Expert Report indicates:

> *"[The] **'safe' dose is referred to by various names**. For the US Environmental Protection Agency (the "EPA"), this dose may be called a 'Reference Dose' **(RfD)** for oral exposures or a 'Reference Concentration' **(RfC)** for inhalation exposures. 'National Ambient Air Quality Standards,' or **'NAAQS,'** are another set of standards set by the EPA that quantify when pollutants may be 'considered harmful to public health and the environment.' NAAQS are generally considered similar to RfCs. The determination of these 'safe' doses or concentrations through dose response assessments is important **since thresholds for adverse effects in the dose or concentration are expected to exist**"* [emphasis added][25]

Although Dr. Maier groups them together, US EPA's basis for establishing RfDs and RfCs is completely different than the basis for establishing NAAQS levels. As described below, RfDs and RfCs are established using thresholds identified (quantified) in animal studies; NAAQS are established using data from human population studies whose effect thresholds change as the exposure-dose-response continuum changes (i.e., studies of populations with reduced air pollution concentrations can identify new/lesser thresholds for responses previously unidentifiable due to the absence of lesser exposed populations).

The US EPA derives chemical-specific RfDs and RfCs to represent an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral (or inhalation) exposure to the human population (including sensitive subpopulations) that is likely to be without risk of deleterious noncancer effects when exposed over a lifetime.[26] Derivations are based upon extrapolation of animal data to anticipated responses in humans: the no-observable adverse

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[24] Brook, R.D., *et al.* Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 121(21):2331-78, 2010.

[25] Andrew Maier. Expert Report of Andrew Maier. Oakland Bulk & Oversized Terminal, LLC v. City of Oakland. Civil Action No. 16-07014(VC), October 6, 2017., p.9, including citation to https://www.epa.gov/criteria-air-pollutants/naaqs-table.

[26] US EPA. Integrated Risk Information System (IRIS) Glossary. Terminology Services. Last updated August 31, 2011. Available from: https://iaspub.epa.gov/sor_internet/registry/termreg/searchandretrieve/glossariesandkeywordlists/search.do?details=&vocabName=IRIS%20Glossary. Accessed: October 14, 2017.